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Bartter Syndrome Masquerading as Acute Kidney Injury in a Neonate

Infants and children with Bartter syndrome present with polyuria and polydipsia, whereas older children present with constipation, salt craving and muscle cramps. The symptomatology is mainly due to renal concentrating defect [1]. This disorder is characterized by hypokalemia, hypochloremia, hypercalciuria, salt wasting with metabolic alkalosis.

A 10-day-old male child born out of third degree consanguineous marriage presented with severe respiratory distress.The antenatal history was uneventful. The neonate was suspected as late-onset sepsis and appropriate management was started. The investigations showed normal counts, C-reactive protein (CRP) levels and urine examination.The condition deteriorated further and required mechanical ventilation. The baby was started on intravenous piperacillin-tazobactam and amikacin, but antibiotics were stopped after 7 days as blood culture sensitivity was negative.

The baby's condition gradually improved and was weaned from the ventilator after 8 days. Renal parameters, urine output and leucocyte counts were monitored regularly and remained normal. The blood gases were all normal. The child was started on breast feeds on day 32 of life and was under observation for proper feed establishment and weight gain. On day 48, the infant developed decreased urine output along with respiratory distress for second time. Investigations showed normal leucocyte counts and normal CRP levels but renal parameters were suggestive of intrinsic renal failure. Peritoneal dialysis and non-invasive ventilation were started. The condition of the child improved and he was weaned from ventilator after 4 days. The renal parameters normalized after 20 cycles of dialysis.Blood and urine cultures were negative.Post-dialysis the child developed polyuria with a daily urine output >8 mL/kg/day. The infant continued to have polyuria inspite of measures to decrease urine output. The infant developed metabolic alkalosis despite acute kidney injury and polyuria. The blood pressures were in normal range. The urine examination showed, red blood cells, granular casts and proteinuria. Urinary electrolytes values showed urine osmolality – 133.2 mOsm/kg (normal 500 – 850 mOsm/kg), urinary chloride–66 mEq/L (normal <10 mEq/L), and spot calcium creatinine ratio - 2.96:1.0 (normal <0.86:1). Serum calcium,vitamin D and parathyroid hormone levels were within normal range. Ultrasonography of kidney and bladder showed calcifications in apex of medullary pyramids suggesting bilateral medullary nephrocalcinosis. We diagnosed our case as type 2 Bartter syndrome.

The classical Bartter syndrome (type 3) is perinatal in onset and presents with polyhydramnios, neonatal salt wasting and recurrent episodes of dehydration. Antenatal Bartter syndrome (type 1,2 and 4) typically manifests in infancy with severe phenotype compared to the classical syndrome [2]. The biochemical features reflect defect in sodium, chloride and potassium transporter on ascending limb of loop of Henle [3].

Various genes are associated with Bartter syndrome [4]; *MAGED2* mutation described recently is associated with transient Bartter syndrome which starts antenatally with severe phenotype and usually resolves by six weeks of age. Our case presented at around six weeks with acute kidney injury without hypomagnesemia [5]. The diagnosis of Bartter syndrome in neonate or infant is suggested by severe hypokalemia, hypochloremia and metabolic alkalosis. Hypercalciuria is typical and nephrocalcinosis is seen resulting from hyper-

calciuria in type 1 and 2. Hypomagnesemia is seen in minority. Urinary levels of chloride are also very much elevated which helps in differentiating this picture from chronic vomiting and cystic fibrosis. The tubular defect in Bartter or Gittlemann syndrome cannot be corrected [6], but with careful fluid and electrolyte management, long term prognosis is good. We treated the child with proper fluid and electrolyte correction following which hyperkalemia improved. The potassium levels normalised after a period of eight days without any therapy for potassium corrections except for restriction. Urinary electrolytes continued to remain elevated. The child was discharged in a stable condition after establishing oral feeds.

The child followed-up with us two weeks after the discharge which was uneventful. Our case focuses light on the rare presentation of Bartter syndrome with acute kidney injury probably due to nephrocalcinosis which might have started in utero.

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Fetal Ovarian Cyst Managed Laparoscopically in the Neonatal Period

Most antenatally diagnosed fetal cystic lesions are of renal or ovarian origin, and timely postnatal diagnosis facilitates early and appropriate management. We report early diagnosis of a fetal abdominal cyst with successful laparoscopic management.

A 1900 gram female baby was born vaginally at 38 weeks to a 24-year-old second gravid mother who had conceived spontaneously. Antenatal period was uneventful. Sonography at 30 weeks of gestation revealed a large well defined intra-abdominal fetal cystic lesion extending from pelvis to sub-hepatic region measuring, 4.8 cm × 4.2 cm × 4.8 cm with evident septations, with maximum wall thickness of 3.5mm. No subsequent antenatal scans were available. Baby did not need any resuscitation after birth but was detected with a palpable lower abdominal lump that was cystic in consistency. Rest of the examination including vitals was normal. A postnatal abdominal sonography showed a large cystic mass located in the right flank extending from sub-hepatic region to the pelvis measuring approximately 6.1 cm × 4 cm × 4.6 cm in size with internal solid areas (possibly fibrinous products) with no obvious vascularity or fluid debris level. Right ovary was not visualized, right kidney was seen distinctly separate from the cyst, and uterus, left ovary and left kidney were normal. Plain *X*-ray abdomen revealed displacement of bowel loops to left side. These findings were consistent with the diagnosis of ovarian cyst with internal hemorrhage (complicated). A thyroid scan performed later was normal. Laparoscopic excision of cyst with preservation of rest of the ovary was performed using three ports and a maximum of 10mm pneumo-peritoneum on day 8 of life. The cyst was seen to originate from right ovary, had a short pedicle and had undergone torsion on its own axis. Dark brown color fluid was aspirated from cyst, which was excised with Harmonic as energy source. Histopathological examination of excised cyst revealed complicated ovarian cyst with necrosed wall. Left ovary was normal. Intraoperative and postoperative course was uncomplicated. Breastfeeding was started on first postoperative day. The baby is currently on follow-up, is feeding and growing normally.

Fetal cystic masses in females are mostly benign and ovarian in origin. In a case series of 41 fetal abdominal cysts, 21 were ovarian cysts whereas 11, 6 and 3 cases were found to be bile duct cyst, intestinal duplication and mesenteric cysts respectively [1]. An antenatally detected isolated, non-lethal lesion should be monitored with repeated ultrasound examination, as the evolution of such a lesion *in utero* is extremely variable [2]. Serial antenatal ultrasounds help to determine the location and nature of the cyst and plan management. Accurate delineation of the mass may require fetal MRI.

Ovarian cysts are the commonest ovarian tumors in newborn period. Simple ovarian cysts are characteristically